DCIEM Research Paper No. 83-R-23

INNER EAR DECOMPRESSION SICKNESS IN THE SQUIRREL MONKEY: OBSERVATIONS, INTERPRETATIONS, AND MECHANISMS

J. P. Landolt, K. E. Money, M. W. Radomski, R. G. Riusech, P. C. Odell, and W. J. Watson

The insidious nature of inner ear decompression sickness (DCS) during deep simulated dives is now well documented in the squirrel monkey (*Saimiri sciureus*) (1–3). In provocative ascents from 274 metres sea water (msw) while breathing a heliox gas mixture, the signs of DCS appear suddenly, mainly in the form of a vigorous head nystagmus, which occurs between 62 msw and the surface. If these monkeys are sacrificed a few days after their dive, the inner ear spaces are usually congested with blood and blood proteins. In those monkeys sacrificed 20 or more days after sustaining an inner ear *hit*, the most common pattern is that of ectopic new bone growth or the presence of fibrous material or both in the fluid spaces of the vestibular apparatus, particularly in the semicircular canal spaces. Damage to the central nervous system (CNS) in the centers subserving vestibular and auditory functions was found only when there was brain damage elsewhere as a result of a severe, generalized DCS.

The purpose of the present report is to summarize the main findings of our research in the area of inner ear DCS in deep diving environments. From these observations, a plausible theory has been developed which explains many of the results that were obtained. Furthermore, interpretation of the results suggests what procedures constitute acceptable therapeutic treatment for the successful management of inner ear decompression sickness.

MATERIALS AND METHODS

Animal Model

Male squirrel monkeys, free from ear infections and other disorders, were used in the study. On the day before the dive, both ear drums were surgically

perforated under anesthesia so that the animal could equilibrate the middle ear with ambient pressure. Functional testing of the vestibular system was performed before and at selected intervals after the dives until the animal was sacrificed for histological study (*see* Ref. 3 for further details). We processed the temporal bones (horizontal sections, 20 μm thick) using the method of Igarashi (4); brain sections were similarly prepared (coronal plane, 20 μm thick).

Decompression Procedures

All experiments were performed in a small animal chamber (Bethlehem Corp., 0.173 m^3 capacity). The dive commenced with air to 13.9 msw; then with helium at a rate of 32.6 msw/min to a depth of 274 msw. After 1 min of bottom time, decompression began at a rate of 18.3 msw/min to 61 msw; then in steps of 6 msw every 4 min to the surface. Throughout the dive, the oxygen partial pressure was maintained at 50.7 kPa (= 0.5 atm).

Hearing Tests and Training

To assess for possible cochlear damage, we tested the hearing in a number of animals, both before and after the dive. The animals were trained by a shock-avoidance procedure to respond to tones. Predive hearing thresholds were determined for a range of frequencies in each animal. The presentation of tones was controlled and delivered by a computer-based system (PDP 11/04 computer; Digital Equipment Corp.). During training and testing, the animals were isolated in a sound-proof booth (Industrial Acoustics Corp.).

After the dive, when the animals had recovered sufficiently, we again tested their hearing to obtain postdive hearing thresholds. These tests were conducted on a regular basis until the animal's audiogram showed no further change, whereupon the animal was sacrificed for histology.

Hyperbaric Oxygen Therapy

With some animals, a regimen of hyperbaric oxygen therapy was instituted immediately after the dive. These animals were treated for three successive days with the U.S. Air Force modification of the U.S. Navy Table 6 Treatment (5) for DCS. Initial trials with control animals had shown that the monkeys could withstand the Table 6 treatment without showing visible signs of oxygen toxicity.

RESULTS AND DISCUSSION

At the time of this report, some 250 monkeys have received inner ear hits in experiments using this diving profile at the Defence and Civil Institute of Environmental Medicine (DCIEM). Table I lists the distribution of new bone

TABLE I

Distribution of New Bone Growth and Fibrous Material in Squirrel-Monkey Semicircular Canals*

Monkey	Depth of Initial Hit on Ascent (msw)	Postdive Survival Time (days)	New Bone Growth and Fibrous Material					
			Left Labyrinth			Right Labyrinth		
8	*12.2*	*38*				*ASC(p)*		
24	61.9	379				*ASC(p),*	*LSC(p),*	*PSC(p)*
32	18.3	211				*ASC(p),*	*LSC(p),*	*PSC(p)*
33	6.1	93				*ASC(p)*		
36	6.1	184	*ASC(e,p)†,*	*LSC(p),*	*PSC(e,p)†*			
51	12.2	56		*LSC(p),*	*PSC(p)*	*ASC(p),*	*LSC(p),*	*PSC(e,p)*
52	36.6	126		*LSC(p),*			*LSC(p)*	
62	24.4	383						*PSC(p)*
68	30.5	290	*ASC(p)†,*	*LSC(p),*				
103	6.1	637		*LSC(p),*				
117	18.3	147	*ASC(e,p)†,*	*LSC(e,p)†,*	*PSC(e,p)†*	*ASC(e,p)†*		*PSC(e,p)†*
119	18.3	133	*ASC(e,p),*	*LSC(p),*	*PSC(e,p)†*			
135	18.3	388	*ASC(p),*	*LSC(p),*	*PSC(p)*	*ASC(e,p),*	*LSC(e,p)†,*	*PSC(e,p)*
136	30.5	213	*ASC(p)*		*PSC(p)*	*ASC(p),*	*LSC(p),*	*PSC(p)*
145	7.6	161	*ASC(e,p)†,*	*LSC(e,p)†,*	*PSC(p)*			
147	30.5	188			*PSC(p)*			
160	30.5	65	*ASC(p),*	*LSC(p),*	*PSC(p)*			
166	18.3	70				*ASC(p)*		
169	12.2	20	*ASC(p)†,*	*LSC(p)†,*	*PSC(p)*			
183	11.3	490	*ASC(p),*	*LSC(p),*	*PSC(p),*	*ASC(p)*		
[illegible]	6.1	353	*ASC(e,p)†*	*LSC(p),*	*PSC(p)*			
	12.2	21				*ASC(p),*	*LSC(p),*	*PSC(p)*
	18.3	197	*ASC(p),*	*LSC(p),*	*PSC(p)*	*ASC(p),*	*LSC(p)*	
[illegible]7	30.5	185	*ASC(e,p),*	*LSC(p),*	*PSC(e,p)*	*ASC(p),*	*LSC(p),*	*PSC(p)*
[illegible]11	18.3	661	*ASC(p),*	*LSC(p)†,*	*PSC(p)*			
Pancho	6.1	239	*ASC(e,p)†,*	*LSC(p)†,*	*PSC(e,p)†*	*ASC(p),*	*LSC(p),*	*PSC(e,p)*
Manuel	30.5	333		*LSC(p)†,*	*PSC(p)*	*ASC(p),*	*LSC(p)†,*	*PSC(p)*
Juan	30.5	178	*ASC(p),*	*LSC(p)†,*	*PSC(p)*			
Enrico	6.1	332	*ASC(p)*					

ASC, *LSC*, and *PSC* signify the anterior, lateral, and posterior semicircular canals; *e* and *p*, the endolymphatic and perilymphatic spaces, respectively. *Information for *Monkeys 8–68* inclusive has appeared previously in Tables 4 and 5 of Ref. 3 (by permission of the American Physiological Society). †New bone growth and fibrous material were also found near ampulla and utricle in perilymphatic space.

growth and fibrous material that has been observed in the semicircular canal spaces in 29 of these monkeys, which were sacrificed between 20 days and 661 days after inner ear DCS. (The complete absence of ectopic new bone growth or fibrosis, or both, in the vestibular spaces has been observed in only seven hit monkeys with these long-term survival times.) *Monkeys 119* and *145* were not recompressed after receiving a vestibular hit, but were brought to the surface slowly. *Monkeys 135, 160, 166,* and *169* were recompressed to depths that eliminated all behavioral signs of vestibular dysfunction before being brought to the surface slowly, at the rate of 0.3 msw/min. The remaining 23 monkeys listed in Table I were compressed to twice the depth at which the hit occurred and then brought to the surface at the slow rate indicated before. Starting immediately after receiving their vestibular hit, *Monkeys 201* and *207* were subjected to a Table 6 treatment for three successive days.

Monkeys Pancho, Manuel, Juan, and *Enrico,* who had been conditioned to discriminate between tones and no-tones, were tested for hearing thresholds before, and at selected intervals after, their dives.

Of the 34 monkeys with long-term survival times that received some form of the recompression treatment, 29 had no apparent behavioral signs of inner ear DCS (such as nystagmus and/or unsteadiness) upon completion of the recompression schedule (including 6 of those in which there was no evidence of bone growth in the canal spaces). However, about one-third of these monkeys developed a positional nystagmus the following day, a clear indication of continuing vestibular dysfunction.

Regardless of whether or not the animal received the recompression or hyperbaric oxygen treatment(s), or both, the entries in Table I indicate the same outcome, i.e., the gross infiltration of fibrotic tissue or new bone, or both, in the canal spaces, as a consequence of inner ear DCS (*cf.* Figs. 1 and 2). (In animals sacrificed shortly after their dive, the manifestations of vestibular-apparatus damage appear in the form of severe hemorrhage in the semicircular-canal perilymphatic spaces, and as blood-protein exudates, mainly, in the endolymphatic spaces; in particular, as an agglutinate to the cupula of the crista ampullaris [3].) This new bone continues to grow slowly until it also occludes the endolymphatic space of the semicircular canal in some cases (*see* (*e,p*) entries in Table I); in others, it continues until it encroaches on the perilymphatic space of the ampulla (*see* * entries in Table I). In either case, the likely result is the same: a progressive lessening of vestibular function, until total malfunction of the involved duct(s) can occur. The table entries also illustrate that this bone growth occurs 1.5 times more often in the left labyrinth than in the right. Furthermore, it may occur in one or more canals on the same side of the head, or it may occur in one or more canals on both sides of the head. Any and all combinations are possible.

It appears that this new bone growth is caused, partly, by a ripping or irritation of the endosteum, which lines the inside of the bony semicircular canals (3). There is also convincing evidence that rapid decompression can generate forces of a magnitude sufficient to fracture the hard temporal bone

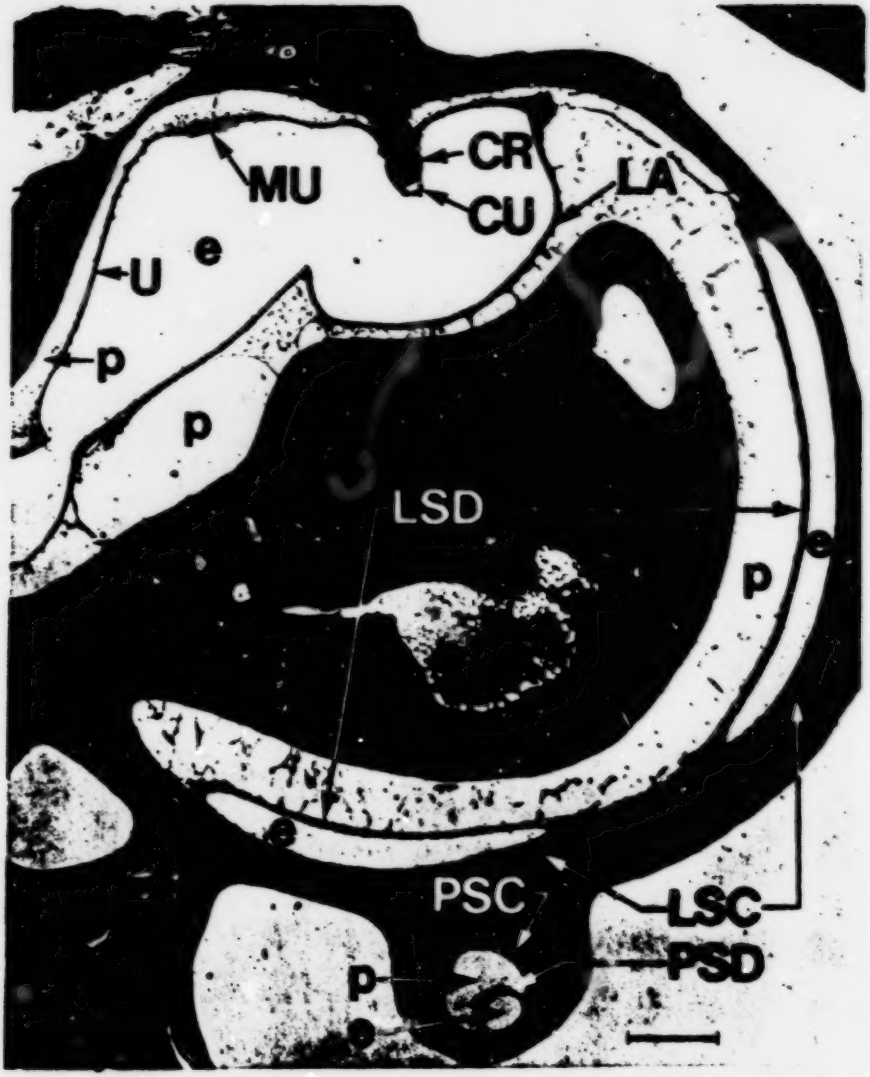

Fig. 1. Normal posterior (*PSC*) and lateral (*LSC*) semicircular canals and associated structures in a horizontal section from the right labryinth of *Monkey 166*. The membranous posterior (*PSD*) and lateral (*LSD*) semicircular ducts lie within the respective canals. *Abbreviations*: *MU*: macular utriculi, the sensory end organ for the detection of linear accelerations and gravity, which is located in the utricle (U); *CR*: crista ampullaris, the sensory end organ for detecting angular accelerations; *CU*: cupula, which interfaces with *CR* and normally fills the ampullary space in its plane of projection, but has shrunk as a result of tissue fixation; *LA*: ampulla of *LSC*; *e* and *p*: endolymphatic and perilymphatic fluid spaces, respectively. The dark half-circular band on the distal surface of the crista is its sensory epithelium. *Bar* = 50 μm.

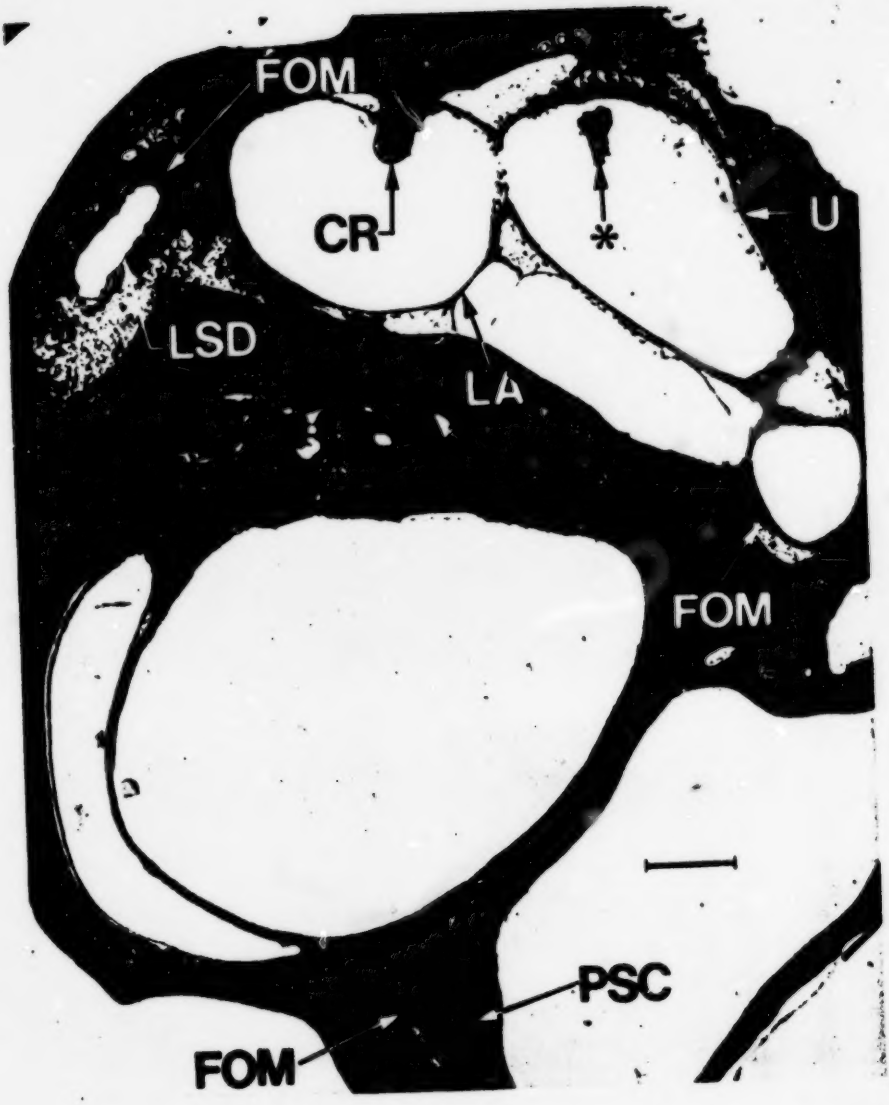

Fig. 2. Horizontal histological section from the left labyrinth of *Monkey Pancho*, illustrating extensive bone and fibrotic growth (*FOM*) in the otic fluid spaces in the posterior (*PSC*) and lateral semicircular canal (*LSD*, *LA*) systems. Note that the cupula is missing and that the "bald" crista ampullaris (*CR*) has only a very thin band of sensory epithelium. This clearly indicates a nonfunctioning sensory end organ. Both otic fluid spaces in the *PSC* have been blocked by FOM, rendering that canal nonfunctional also. *Symbols*: *U*: utricle; *: designates otoconial (ear-stone) mass that has been displaced from the macula utriculi (which is not clearly defined in this photo). *Bar* = 50 μm.

that is contiguous to the bone comprising the semicircular canal, as well as the canal wall itself (6).

In an investigation of the biophysical mechanisms responsible for causing this type of damage in monkeys during rapid decompression, Ward and his colleagues (7) have produced a model which theorizes that, for certain conditions during decompression, large stresses can be produced by bubble nucleation and growth within osteoclast cell cavities in the bone, such as are found in temporal and canal bone. Bubble nucleation is thought to occur within the conical processes of the "ruffled" border—a specialized membrane involved in bone resorption—of an osteoclast cell. This theory predicts that the essentially incompressible cytoplasm in the bone cell (which forms a constant-volume, basically closed cavity of liquid-gas solution) experiences a pressure rise with bubble growth sufficient to produce a force capable of fracturing the bone in which it is contained. Near the surface during ascent, the fluid spaces of the semicircular canals would be at ambient pressure compared to the very high pressure that would be experienced by the cytoplasm within the osteoclast cavity during bubble growth, after exposure to 274 msw. Therefore, once a critical state is achieved, the bubble would grow, causing a large transient increase in pressure relative to that of ambient pressure. Accordingly, this would result in a sudden implosive fracture of the (presumably) rigid temporal and canal bone into the semicircular canal spaces during the final phases of decompression. This theory is consistent with some of the types of semicircular canal damage that have been observed histologically after rapid decompression (6). Venter and his colleagues (8) have found that a mean pressure of 1.6 ± 0.4 MPa is required to fracture the full thickness of the semicircular canals in squirrel monkeys; this pressure is consistent with that predicted by Ward's theory.

The implosive force also causes a bolus of pressure wave energy to move rapidly along the canal; this energy could tear the endosteum and loosen the membranous semicircular ducts from their anchorage to the canal wall as well as provide a substantial transient stimulus to the vestibular end organs. This transient stimulus likely explains why a vestibular hit appears suddenly during decompression in most of these cases. (Vestibular hits resulting from blood supply changes to the vestibular end organs and from central vestibular dysfunction can also appear suddenly.)

The theory of Ward and his colleagues (7) also predicts that, under similar conditions, for less severe pressure, bubble nucleation and growth in bone cells will not produce the pressure required to break the bone. However, this pressure difference, which could be of the order of 0.5 MPa, would still damage or kill the bone cells by mechanical distortion. This mechanism could explain the empty bone cell cavities found in the long bones of divers suffering from aseptic bone necrosis, a debilitating condition that becomes progressively more evident several years after undergoing decompression procedures.

It is instructive to compare the manifestations and symptoms of inner ear DCS in the squirrel monkey with those observed in the commercial diver. In

particular, the clinical observations on eight divers by Komordin (9) are pertinent and characteristic. The sickness appeared suddenly during decompression, at depths of 40–45 msw, 1 ½ to 3 h from the time the dive began. In all cases, the bottom depths exceeded 150 msw, and the breathing gas mixture was oxy-helium. Severe vertigo, nystagmus, nausea, emesis, tinnitus, a loss of spatial orientation, and a decrease in hearing were evident in most cases. Symptoms associated with the decompression syndrome, such as joint pain or itching of the skin, were usually absent. Immediate recompression was successful in only about a third of these instances, and then only under the conditions of high pressure for long periods.

We have recently completed the histological study of the temporal bones of a professional diver who died (of unrelated causes) 56 days after sustaining a severe inner ear hit to his left labyrinth after a dive to about 100 msw for 19 min on trimix (10% N_2). Some 6 hours after the dive, during sleep, this diver experienced dizziness and knee pain and was, therefore, promptly recompressed at least twice (one for 52 h) in an attempt to provide relief (unsuccessfully). Clinical tests indicated a total loss of vestibular function and a partial hearing loss in the left ear. The histological study revealed ectopic new bone growth and fibrosis in one of the semicircular canals of the left ear (report in preparation, 1983). Clearly, many of the manifestations and symptoms of inner ear DCS in man and monkey are of a similar nature.

The insidious nature of the vestibular lesions makes it imperative that any diver who has experienced inner ear DCS should obtain follow-up clinical evaluation over a period of several years before a clean bill of health is given. The fact that many divers experience vertigo during decompression suggests that pathological bone growth may, perhaps, be quite common in older divers. (It would be interesting to know whether or not divers hear a click, snap, or bang preceding a vestibular hit, as might be expected on the basis of Ward's model [7].) The slow growth rate of this ectopic bone would be expected to give CNS compensatory mechanisms time to develop and restore normal balance during ambulatory situations when there is good visibility. However, exposure to conditions of neutral buoyancy and poor visibility, such as can occur while diving, could lead to disorientation and may threaten the life of a diver who had previously received such a hit.

Accordingly, it may be prudent for every diver who has sustained such a hit to obtain either a temporal-bone computerized tomographic scan (10), or, better yet, one that utilizes nuclear-magnetic-resonance imaging techniques (since this does not require exposure to radiation but provides similar information) to assess the true nature of the damage before returning to diving. Moreover, older divers, even if they have not experienced a direct vestibular hit, should routinely obtain a thorough otoneurological evaluation (complete with scan) because there is some evidence that a series of subthreshold vestibular assaults could produce a similar pathology.

The clinical practices of the French specialists in the treatment of decompression-caused ear injuries in divers are of interest to this study (11–14). Regardless of the dive, whether on compressed air or helium, shallow or deep

saturation, the recommended practices appear to be the same: the treatment consists of hyperbaric oxygen therapy in combination with vasodilators, corticosteroids, and heparin in small doses. Experience has shown that hyperbaric oxygenation with this type of adjuvant drug therapy is very effective in reversing peripheral cochlear dysfunction. However, the French have noted that, in spite of immediate treatment, peripheral vestibular lesions from dives to great depths can be severe from the onset and, furthermore, may be permanent (15) and worsen with time (16). McCormick et al. (17) have recommended the prophylactic use of heparin and Novotny (18) the use of nicotinic acid, a reputed vasodilator, for ameliorating decompression-induced hearing loss.

Given the nature of the damage sustained by the peripheral vestibular apparatus during inner ear DCS, it is not surprising that neither hyperbaric oxygen therapy (*Monkeys 201* and *207* listed in Table I) nor prompt recompression were found to be beneficial forms of therapy. Similarly, the beneficial effects of adjuvant drug therapy with vasodilators, anti-inflammatory agents, and anticoagulants would likely be extremely limited. Diazepam, a tranquilizer and anticonvulsant agent, which has been recommended as adjuvant therapy for labyrinthine DCS (19), would also seem to be powerless to either reverse or prevent this kind of damage, though it may provide some much-needed relief from vertigo.

Because the nature of the damage to the peripheral hearing organ in squirrel monkeys is quite different from that to the vestibular apparatus, it may be that hyperbaric oxygen therapy as used by the French (11–14) and others (9,18) could be a beneficial form of treatment. Indeed, studies to date in our laboratory have shown that the cochlear damage following a successful hit first appears histologically only in the form of a blood-protein exudate and an occasional hemorrhage of the cochlea. Therefore, it is important that the highly specialized receptor hair cells, which consume large amounts of oxygen, do not become anoxic as a result of blood interruption to the organ of Corti. (It bears mention that the organ of Corti always appears intact and functional in celloidin histological sections.) In animals sacrificed several months after receiving an inner ear hit, the cochlear fluid spaces have become quite clear, containing much less exudate than is observed shortly after the dive (Fig. 3). In this regard, the amount of exudate is similar to that observed in control animals. Moreover, bone or fibrotic growth, or both, have never appeared in the cochlea. Notwithstanding the cochlear deficits, gradual recovery of some hearing function was evident in monkeys that were tested behaviorally for hearing loss (Fig. 4), even though there was strong evidence that vestibular function remained severely interrupted (e.g., *Pancho*, *Manuel*, *Juan*, and *Enrico* listed in Table I). Of course, the fluid spaces in the inner ear are shared by both the vestibular apparatus and the cochlea. Thus, injury to one organ often results in a decrement of sensory function in the other organ. In this regard, the hemorrhage or blood vessel blockage that may result from bubble nucleation and growth in the ear vessels during decompression can be significant. The effect on the fluids in one organ may cause changes in the

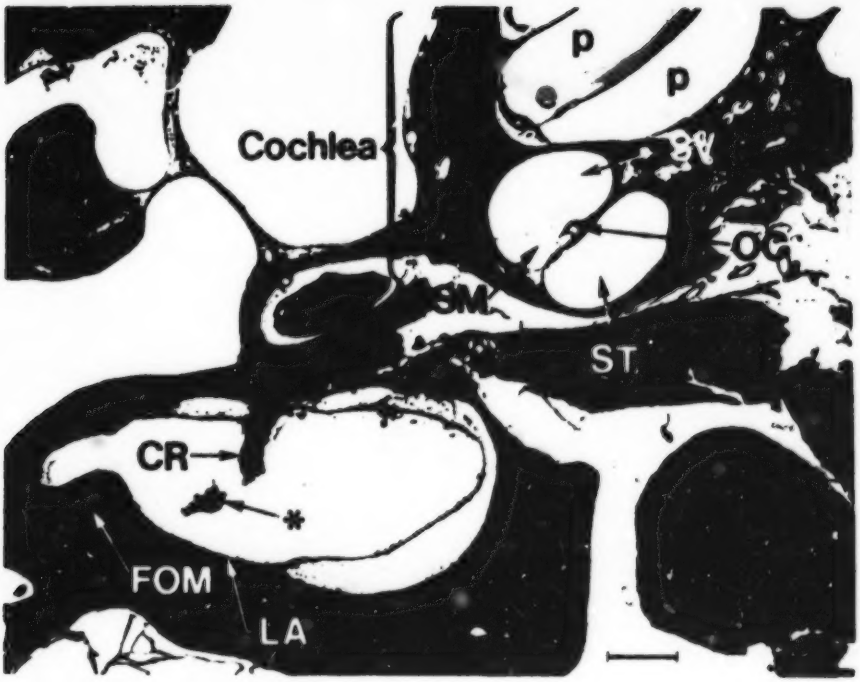

Fig. 3. Horizontal histological section illustrating the presence of a normal-appearing cochlea next to a pathological lateral ampulla (*LA*) from the left labyrinth of *Monkey 169*. Both the endolymphatic (*e*) and perilymphatic (*p*) spaces in the cochlea are clear, and the organ of Corti (*OC*), the sensory end organ of hearing, appears normal. The presence of fibrotic material (*FOM*) in the perilymphatic space of the *LA* is evident. *Abbreviations*: *SV*, *ST*, *SM*: scalae vestibuli, tympani, and media, respectively (otic fluid spaces in the cochlea); *CR*: crista ampullaris. The * identifies cupular remnants that have become detached from the *CR*. *Bar* = 50 μm.

electrolytic and protein compositions in the other organ, resulting in an interference with the physiological mechanisms involved in normal sensory function. Presumably, as the initial disorder subsides and clears, the mechanisms which control sensory function are re-established. This might explain why, sometimes, there is sensori-neural hearing loss without any apparent concomitant pathological basis.

All of the hits that are recorded in Table I occurred at various depths during ascent and never at the surface. There are, however, many instances of human divers whose symptoms appear suddenly, only after decompression has been completed. This could signify that there are gas bubbles that are trapped in the conical processes, but whose further growth has been inhibited by the rigid surrounding bone. It is possible that such bubbles could remain quiescent

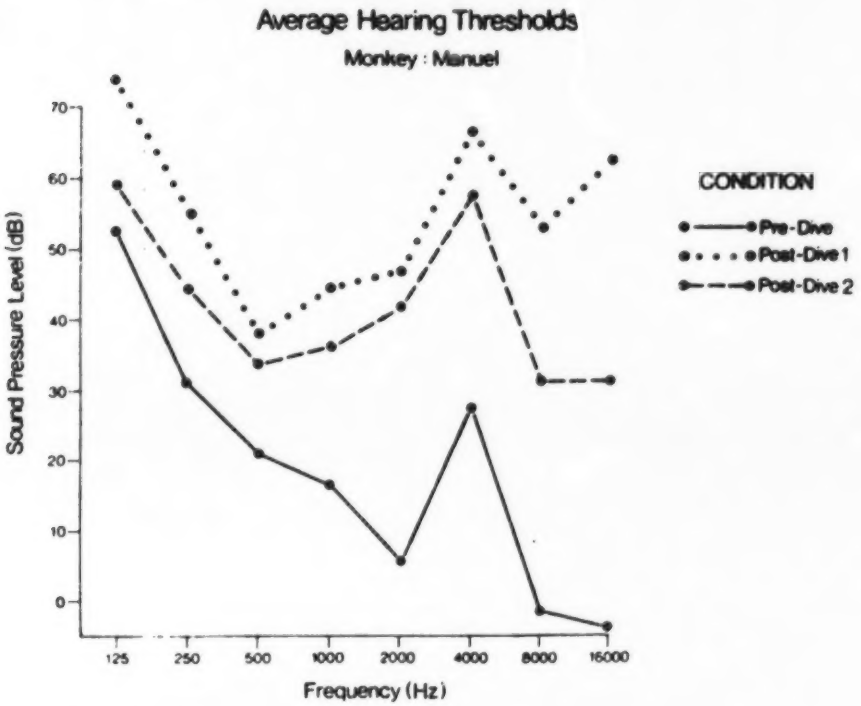

Fig. 4. Curves of average hearing thresholds, in decibels (dB), as a function of the stimulus frequency for *Monkey Manuel*. The *conditions* are: *Predive*, averaged over 3 tests; *Postdive 1*, 92–125 days after the dive, averaged over 4 tests; and *Postdive 2*, 287–301 days after the dive averaged over 4 tests. As indicated, there was some recovery of hearing between the *Postdive 1* tests (when the hearing threshold was at its worst) and the *Postdive 2* tests (the final tests before the animal was sacrificed 32 days later). The *curves* indicate that a (possibly permanent) residual hearing deficit of 10 dB (at the lower frequencies) to 30 dB (at the higher frequencies) remains at the time of sacrifice.

and asymptomatic, until some unknown, cataclysmic "jolt" causes them to continue to grow and, thereby, fracture the bone containing them. Accordingly, a prudent method for preventing this type of subsequent bubble growth (and a treatment that probably would benefit the cochlea) might be the mandatory use of the U.S. Air Force modification of the U.S. Navy Table 6A Treatment (5) for gas embolism whenever DCS is suspected. It bears mention here that the only effort at this laboratory (DCIEM) with the modified Table 6A Treatment was equivocal. *Monkey W13* (not mentioned previously), upon receiving a CNS hit at 30.5 msw on ascent from a 274-msw dive, was recompressed to 50 msw. This treatment removed the CNS hit but left the monkey with an apparent vestibular hit (possibly in the vestibular central

pathways), which persisted until the animal was (slowly) decompressed to 19.5 msw, whereupon all signs of the hit disappeared. On reaching surface, *Monkey W13* was given a single modified Table 6A Treatment. This monkey was sacrificed 86 days after the dive; subsequent temporal bone histology indicated that the vestibular organs were intact and in good condition.

Farmer (19) has indicated that a drug treatment which relies upon increasing inner ear blood flow, as has been recommended by the French and others (11–14,17,18), may result in additional bleeding; or, it might cause blood flow to be shunted to more peripheral regions, thereby counteracting its intended purposes. Such agents are considered by Farmer to be potentially harmful and are not recommended. The results from this study would tend to support Farmer's reasoning.

SUMMARY AND CONCLUSIONS

As a result of our research on the nature of the inner ear DCS resulting from deep dives, the following statements can be made:

1) The sickness occurs very suddenly during the ascent phase of the dive (between 62 msw and the surface for squirrel monkeys on a 274-msw dive).

2) Prompt recompression appears to lessen (or even eliminate) the acute behavioral signs and symptoms of inner ear DCS; it does not, however, reverse the pathological damage to the vestibular apparatus that is provoked by the dive.

3) Serial histological sections of the brains and temporal bones of monkeys which had received inner ear hits show that, if the only symptoms are of a vestibular nature, then the problem is likely to be only in the ear and not in the brain.

4) Cochlear damage first appears in the form of blood and blood-protein exudates in the otic fluid spaces; much later, these spaces appear clear, similar to those observed in control animals. Hearing tests show that some of the monkeys gradually recover some of their hearing deficit (without benefit of adjuvant drug therapy); however, a residual loss remains permanently.

5) Vestibular-apparatus damage first appears in the form of severe hemorrhage and blood-protein exudates in the otic fluid spaces; later, these spaces become invaded by ectopic new bone growth and fibrous material.

6) Histologically, temporal- and canal-bone breaks are prevalent in some of the hit monkey ears. A theory was developed (7) which predicts that such breaks could occur from "imploding" forces that are caused by the large pressures (1.6 MPa or more [8]) that are generated as a result of bubble nucleation and growth within osteoclast (bone) cells during decompression.

7) Cochlear lesions likely occur as a result of bubble formation and growth within microvessels, and their consequent blockage or rupture, or both, causes hemorrhage or blood-protein exudatation, or both. Because of the damage of anoxia to the highly specialized receptor hair cells of the cochlea under such conditions, immediate hyperbaric oxygen treatment would appear to be a necessary requirement.

8) Vestibular lesions of the type described in this report would not benefit from (or be aggravated by) hyperbaric oxygen therapy, whether the treatment used is for DCS (Table 6 treatment) or for gas embolism (Table 6A treatment). However, since cochlear lesions often appear with vestibular lesions, such treatment should be a recommended practice whenever a cochleovestibular insult is indicated.

9) Adjuvant drug therapy that increases inner ear blood flow is contraindicated in the treatment of inner ear DCS.

10) Any diver who has experienced inner ear DCS should obtain extensive follow-up clinical evaluations before returning to diving. Older divers, whether or not they have received a direct inner ear insult during decompression, should routinely obtain a thorough otoneurological examination.

Acknowledgments

This research was supported, in part, by the USN Office of Naval Research, Contract N00014-77-G-0077. DCIEM Publication Number 83-P-23. The authors thank Mr. Jerry Laufer for the histological preparations, Mr. Alcide Nicholas for general technical help, and Mr. Ron Cardin for editorial assistance with the manuscript.

This work is dedicated to the memory of Dr. Gosta F. Dohlman, whose association with DCIEM enriched us all.

References

1. Landolt JP, Money KE, Topliff EDL, Powers KD, Johnson WH. Vestibulocochlear dysfunction in squirrel monkeys during rapid decompression. Med Aeronaut Spat, Med Subaquat Hyperbar 1977;16:377–381.
2. Landolt JP, Money KE, Topliff EDL, Ackles KN, Johnson WH. Induced vestibular dysfunction in squirrel monkeys during rapid decompression. Acta Oto-Laryngol, 1980;90:125–129.
3. Landolt JP, Money KE, Topliff EDL, Nicholas AD, Laufer J, Johnson WH. Pathophysiology of inner ear dysfunction in the squirrel monkey in rapid decompression. J Appl Physiol: Respirat Environ Exercise 1980;49:1070–1082.
4. Igarashi MA. A standard technique for temporal bone preparation. Pensacola, FL: Naval Aerospace Medical Institute, 1966. (Monograph 13)
5. Kindwall EP. Decompression sickness. In: Davis JC, Hunt TK, eds. Hyperbaric oxygen therapy. Bethesda: Undersea Medical Society, Inc., 1977;125–140.
6. Fraser WD, Landolt JP, Money KE. Semicircular canal fractures in squirrel monkeys resulting from rapid decompression: Interpretation and significance. Acta Oto-Laryngol 1983;95:95–100.
7. Ward CA, Johnson WR, Venter RD, Ho BN, Forest TW, Fraser WD. Heterogenous bubble nucleation and conditions for growth in a liquid-gas system of constant mass and volume. J Appl Phys 1983;54:1833–1843.
8. Venter RD, Ho BN, Johnson WR, Ward CA, Fraser WD, Landolt JP. Fracture studies on semicircular canals of the inner ear. Undersea Biomed Res 1983;10:225–240.
9. Komordin IP. Some peculiarities of Meniere's syndrome in deep sea divers (in Russian). Voennomed Z 1973;5:66–67.
10. Valvassori GE, Mafee MF, Dobben GD. Computerized tomography of the temporal bone. Laryngoscope 1982;562–565.

11. Le Mouel C, Fretillere H, Oliva A, Renon P, Angot A. L'oxygénothérapie hyperbare dans le treatment des accidents cochléo-vestibulaires de la plongée. Med Aeronaut Spat, Med Subaquat Hyperbar 1977;64:385–388.
12. Renon P, Angot A, Suc B. Conceptions pratiques des accidents de l'oreille interne chez les plongeurs. Med Aeronaut Spat, Med Subaquat Hyperbar 1980;74:95–101.
13. Demard F. Variations de pression et oreille interne en plongée. Med Aeronaut Spat, Med Subaquat Hyperbar 1980;74:89–94.
14. Le Mouel C, Suc B, Asperge A. Traitement hyperbare des accidents de l'oreille interne liés à la plongée. Med Aeronaut Spat, Med Subaquat Hyperbar 1981;79:242–246.
15. Demard F, Olivier JC, Paolino M. Evolution et séquelles tardives des accidents cochléo-vestibulaires de la plongée profonde. Med Aeronaut Spat, Med Subaquat Hyperbar 1977;64:389–393.
16. Bertoin R. Evolution clinique des accidents labyrinthiques survenant chez les ouvriers travaillant en air comprimé. Arch Mal Prof 1953;14:221–224.
17. McCormick JG, Philbrick T, Holland W, Harrill JA. Diving induced sensori-neural deafness: Prophylactic use of heparin and preliminary histopathology results. Laryngoscope 1973;83:1483–1501.
18. Novotny, GM. "Cochlear bends." J Otolaryngol 1980;9:395–398.
19. Farmer JC. Inner ear injuries in diving—Differential diagnosis of inner ear decompression sickness and inner ear barotrauma. In: Bachrach AJ, Matzen MM, eds. Underwater physiology VII. Proceedings of the seventh symposium on underwater physiology. Bethesda: Undersea Medical Society, Inc., 1981;805–810.

POOR

QUALITY

ORIGINAL